

For Anne Wagon
with best wishes—
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NOTES ON ANESTHESIA

COLLEGE OF PHYSICIANS AND SURGEONS
COLUMBIA UNIVERSITY, NEW YORK

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FOREWORD

In the absence of an adequate anesthesia textbook, the following notes are offered as a supplement to the references listed on the next page. The student who finds that the physiological mechanisms briefly referred to in this outline are unfamiliar to him will do well to review them at this time in his textbooks, and lecture notes of previous courses.

There are three people of great importance in the conduct of the anesthesia: the patient, the anesthetist and the surgeon. The statements which follow take for granted an ideal situation: an experienced surgeon who works quickly and gently, and leaves the choice of the anesthetic agent and technic and the conduct of the anesthesia to the anesthetist; a wholly capable anesthetist who knows how to choose and use all the anesthetic agents and technics well, and who understands fully the surgeon's needs; and the most important, but also the most variable individual, the patient. He becomes less unpredictable the more the surgeon and anesthetist know about his disease and how it affects him as a whole. The anesthetist cannot adequately evaluate his patient unless he is first a good physician with a working knowledge of physiology.

SUGGESTED READING

1. Guedel, A. E. "Fundamentals of Inhalation Anesthesia"
2. Goodman and Gilman. "Pharmacological Basis of Therapeutics"
First 100 pages, Chaps. 9, 12 and Section V.
3. American Medical Association. "Fundamentals of Anesthesia" 2nd Ed.
4. Cullen, S. C. "Anesthesia in General Practice"
5. Courville, C. B. "Untoward Reactions to Nitrous Oxide Anesthesia"
Excellent for pathology of anoxia.
6. Drinker, C. K. "Pulmonary Edema and Inflammation"
Good background for pulmonary complications.
7. Miller, W. S. "The Lung"
8. Macleod, J. J. R. "Physiology in Modern Medicine"
Chapter on Respiration by Carl F. Schmidt
9. Adriani, John "Pharmacology of Anesthesia"
"Chemistry of Anesthesia"
"Technics and Procedures of Anesthesia"
10. "Anesthesiology," a monthly Journal
11. Gillespie, N. "Endotracheal Anesthesia" 2nd Ed.
12. Leigh, M.D. "Pediatric Anesthesia"

RESPIRATION

It is most important that the anesthetist be thoroughly familiar with the physiology of respiration since the lungs are the only site at which oxygen may enter the body, the chief site of excretion of carbon dioxide, and the site of absorption and excretion of many of the anesthetic agents. The mechanics, physical principles, and physiological control which cause these gases and vapors to enter and leave the lungs should therefore be well understood, in order that the anesthetist may recognize the changes which take place when the normal state passes to the pathological, or when consciousness is replaced by progressively deepening anesthesia. Careful observation of all phases of respiration tells more about the depth of anesthesia than all the other signs combined.

Control

The conscious control of respiration disappears entirely with the onset of the anesthetic state. Respiration becomes machinelike, and variations in it depend entirely on the mechanical or physiological changes produced by the anesthetist or surgeon.

The unconscious or automatic control is chiefly chemical in nature. It is the concentration of the H ions in the respiratory center which affords second to second control of respiration. Because of the great diffusibility of carbon dioxide through cell membranes, it usually causes these changes in pH, but they may be due to lactic acid, unoxidized fatty acids, etc. Hyperpnea follows an increase in acidity and diminished tidal volume follows a decrease in acidity. When the respiratory center is abnormally depressed by drugs (anesthetic agent, morphine, barbiturates, etc.) the site of automatic control is probably shifted to the carotid bodies. A low oxygen tension is now the respiratory stimulant,

not pH changes. Excess oxygen under these conditions will cause a period of apnea, until either the hypoxic stimulus recurs, or the CO_2 tension rises above the level of the respiratory center's threshold.

Reflex control is present to a lesser extent. Common sites of origin for the afferent impulses are the skin (pain, cold), the receptors of the vagus in the respiratory tract, the peritoneum, rectal sphincter, the carotid sinus, aortic arch, and carotid body. The afferent impulse probably travels up the peripheral or somatic nerves to the respiratory center. The efferent impulse may travel down the vagus to the larynx and bronchi to cause laryngospasm or bronchospasm, or down the cord to the cervical region (phrenics) and thoracic regions innervating the intercostal muscles.

It is an error to presuppose a normal respiratory center in the surgical patient. Although sometimes stimulated (pain, fever) it is more often depressed, and its threshold to stimuli raised because of drugs used, or because of abnormal metabolism arising from the patient's illness. In order of frequency, the most common respiratory depressants are the anesthetic agent, the premedication drugs, oxygen lack and a marked CO_2 excess.

The production of oxygen lack, or carbon dioxide retention is as inexcusable as an overdose of premedication drugs or anesthetic agents. A relative oxygen lack (hypoxia) is frequently present with certain diseases such as hyperthyroidism, severe anemia, pulmonary fibrosis and poorly compensated circulatory systems, but the oxygen lack should be treated before operation is considered.

Anatomy

A knowledge of the anatomy of the respiratory tract is important so that we may predict and thereby prevent respiratory obstruction, or locate it promptly should it occur. Respiratory obstruction is the commonest anesthetic complication. Frequent causes for obstruction and their treatment are listed on the next page.

<u>Upper Respiratory Tract</u>	<u>Treatment</u>
Tongue against pharyngeal wall in supine position	Changing position of head; artificial airway
Large tonsils, pharyngeal tumors; pharyngeal edema	Artificial airway beyond point of obstruction
Fluids: mucus, blood, pus, vomitus	Prophylaxis, premedication, head down, suction
Laryngospasm: direct irritation anesthetic agent, mucus, vomitus, pus	Slower induction, use of non-irritating agent first, premedication, properly fitting airway, gravity and suction
Reflex laryngospasm	Gentle surgery; adequately deep anesthesia; avoidance of oxygen lack and CO ₂ excess
Nasal obstruction plus tightly closed mouth: adenoids, rhinitis, weeping	Preoperative shrinkage nasal mucous membrane; oral airway early

Lower Respiratory Tract

Fluids: mucus, vomitus, blood, pus	Prophylaxis; head down, suction
Asthma; reflex bronchospasm	Proper selection anesthetic agent; helium
Substernal thyroid, mediastinal tumors compressing trachea	Airway established beyond obstruction; helium

Mechanics

Contraction of the diaphragm and external intercostals produces the negative pressure which pulls the lungs outwards, causing an inrush of air. Relaxation of these muscles causes the reverse process, expiration. Only in forced expiration are the internal intercostals and the abdominal muscles contracted. Normally there is always a negative pressure in the pleural space. It can become more negative by inspiring against a closed glottis, or by producing a massive atelectasis. It is more positive with forced expiration against a closed glottis (beginning of a cough) and with pneumothorax. The intrapulmonary pressure, in the resting phase of respiration, is the same as atmospheric pressure. Disturbed

intrathoracic pressure relations may embarrass the circulation as well as the respiration by altering the venous return to the heart. Maintenance of the proper pressure relations depends also on an intact thoracic cage, with good mobility at the costovertebral and sternoclavicular joints.

Lung motion is essentially passive. Active changes in bronchial lumen and length may contribute to lung expansion with inspiration and expiration is aided by the elastic recoil of the lung tissue. The best aerated portions are the lower lobes and anterior parts of the upper lobes. With inspiration the apex and lung root move downward, the lung base downward and outward, the remainder of the lung outward and slightly upward.

Numerous factors may interfere with the mechanics of respiration, causing a decrease in vital capacity, and thereby producing inadequate transport of anesthetic gases, oxygen and carbon dioxide to and from the blood stream.

Pathological:

Pulmonary....Fibrosis of interstitial tissue
 Chronic bronchitis; emphysema
 Asthma
 Tuberculosis
 Pneumonia
 Atelectasis
 Pneumothorax
 Empyema
 Pleural adhesions

Skeletal.....Osteoarthritis of spine
 Previous rib resections
 Kyphoscoliosis

Muscular.....Residual poliomyelitis
 diaphragmatic paralysis
 intercostal paralysis
 paralyzed spinal muscles with chest deformity
 Diaphragmatic hernia

Mediastinal..Tumors, substernal thyroid
 Pericardial effusion

Abdominal....Distension of hollow viscera
 Pregnancy
 Ovarian cyst; fibroid uterus
 Ascites
 Hepato-and splenomegalies

Produced during operation:

Change in position: head down, prone, lateral, kidney bridge
 Open chest for thoracic surgery
 Weight of assistant's arm on chest
 Removal of ribs, thoracoplasty
 Retractors on costal margin
 Pads against diaphragm
 Depression of muscular activity by anesthetic drugs
 Atelectasis
 RESPIRATORY OBSTRUCTION

CIRCULATION

Circulation is as important as respiration in the proper distribution of oxygen and the anesthetic agent to the tissues, and in the excretion of carbon dioxide and the anesthetic agent. Circulatory signs are not especially helpful in determining the depth of anesthesia, but are of great assistance in determining the reaction of the patient to the operative trauma and the anesthesia. Only with an accurate and frequent record of pulse rate by palpation, and blood pressure by auscultation can a proper interpretation and prognosis be made.

Control

The control of the circulation is like respiration in that the stimulus is chemical in nature, but unlike respiration, in that the "pacemaker" is in a peripheral region rather than in the medulla. The sino-auricular node is the origin of the heart beat, and changes in acidity of the tissues which make up the S-A node influence the rhythm greatly. In the heart, the important chemical stimuli are the inorganic ions, Ca, K and Na, not carbon dioxide. The conducting tissue of the heart is quite different from the nervous tissue carrying the

respiratory impulses, but in both systems activity leads to a refractory period, when further activation cannot take place.

Several neurological connections exist which contribute to the control of circulation.

a. The right and left vagus nerves supply the S-A and A-V nodes respectively. Their normal action is one which keeps the heart in check. Stimulation of the vagus produces a marked slowing in rate, with or without a heart block.

b. The accelerator nerves, belonging to the sympathetic nervous system, supply the heart much more diffusely. Their cell bodies are located in the thoracic cord, from T1 to T6 and their first synapse occurs in any of the cervical and upper five thoracic sympathetic ganglia. The postganglionic fibers may travel in the cardiac plexus, or actually join the vagus nerve trunk. The sympathetics and vagus tend to balance one another normally, but stimulation of one system depresses the activity of the other, and blocking of one system produces an exaggeration of the signs of activity of the other.

c. Both the divisions of the autonomic nervous system supply the peripheral arterioles. Vasoconstriction occurs with sympathetic stimulation; vasodilation occurs less forcefully but is probably mediated by the parasympathetic system. Exceptions to this general rule are the coronary and cerebral vessels, which dilate with sympathetic stimulation. Capillaries apparently possess contractility quite independent of the state of the arterioles, or neurological connections.

Afferent connections of the nervous system with the circulation exist in many places. The most important follow:

a. Carotid sinus: Receptors are present in the walls of the carotid sinus, at the bifurcation of the common carotid into its external and internal branches. The afferent impulses pass along the carotid sinus nerve, a branch of the glossopharyngeal nerve, and branches of the vagus and cervical sympathetics. Reflexes

from this region normally keep the blood pressure at a constant level. Pressure changes inside or outside the sinus wall are the chief stimulating factors. Overstimulation results in hypotension and bradycardia.

b. Aortic arch: the afferent path is up the vagus nerve to the medulla. Its reactions are similar to the carotid sinus reflexes.

All these impulses are collected by the vasomotor center in the medulla, which synchronizes the circulatory reaction. Changes in respiration frequently occur at the same time because of the close relation of the respiratory and vasomotor centers.

Shock

This is the commonest circulatory accident. The word is poorly chosen, for many connotations have been ascribed to it. What is meant is a discrepancy between the circulating blood volume and the capillary bed. The sequence of events in increasing shock is:

1. Precipitating trauma: severe burn, hemorrhage, prolonged surgical trauma, deep anesthesia
2. Capillary dilatation and arteriolar constriction: trapping of much blood in capillary bed making it unavailable for general circulation
3. Constriction of spleen to supply more blood
4. Increased heart rate, to deliver diminished volume better
5. Diminished venous return
6. Increased respirations (hypoxic stimulus to carotid body) to supply more oxygen to overworking heart, until center damaged by hypoxia
7. Loss of proteins through capillary wall to tissues
8. Viscosity of blood greater - more difficult for heart as a pump
9. Further capillary damage from prolonged hypoxia. Now irreversible
10. Failure of myocardium because of poor oxygenation

It is obvious that treatment of shock must be prompt and rational. It is directed towards:

1. Supplying ample oxygen
2. Restoring blood volume
3. Restoring plasma proteins
4. Restoring RBC, if blood has been lost

The anesthetist's immediate responsibility is to institute means of providing the patient's tissues, especially the cerebral tissues, with ample oxygen, and to maintain optimal oxygenation while fluids, blood and blood substitutes are obtained and administered by others. If help is not available the anesthetist should first set up a source of excess oxygen and an infallible airway, before embarking on what may be a difficult and time-consuming venipuncture in a patient with peripheral vasomotor collapse.

There is no rationale in giving cardiac stimulants. Adrenalin, Analeptics (coramine, metrazol, etc.) and carbon dioxide stimulate heart muscle ONLY if it is well oxygenated.

Anesthetic Implications

1. Evaluation of the risk of a cardiac patient is merely the evaluation of his compensation and reserve. Any surgical operation or anesthesia imposes a strain on his circulatory system, and a careful history of his tolerance of other kinds of strain will give better clues as to the length and severity of the surgical procedure he can tolerate, than physical examination of his heart and peripheral vessels. Determining the presence or absence of murmurs, abnormal rhythms, or vascular thickening cannot alone lead to an accurate prognosis as to functional efficiency and adaptability of the circulatory system.

2. All anesthetic agents depress heart muscle directly, making it a less effective pump. The deeper the anesthesia, the worse is the depression. Chloroform and ethyl chloride are the most severe depressants.

3. Ether anesthesia, carried no deeper than second plane, is probably the

safest for a cardiac patient. Because of its irritating effect on the respiratory system, inspiration is stimulated which causes a better venous return to the heart. Within certain limits, this means an improved circulation.

4. Oxygen lack is the worst depressant for heart muscle especially if it is already damaged. (Myocarditis, rheumatic fever.) Ample oxygen **MUST** be supplied to cardiac patients.

5. The conduction system in the heart is often affected by the agent. Cyclopropane frequently produces irregularities in rhythm which are not permanent. All drugs, even procaine and the barbiturates, cause EKG changes.

6. The vasomotor center is depressed especially by the non-volatile drugs: short-acting barbiturates, avertin. Deep anesthesia with the inhalation agents also causes a marked depression, so that compensation for impending shock, by vasoconstriction, is completely lost.

The anesthetist always attempts to keep the blood pressure and pulse at their normal levels, in spite of the surgery. Following are some of the common reasons for changes in them.

Increased Pulse Rate

Oxygen want, early
Carbon dioxide excess
Hemorrhage
Light anesthesia
Ether
Excessive sweating
Increased adrenalin output
Fluids given too fast
Analeptics
Large doses atropine or scopolamine

Decreased Pulse Rate

High spinal anesthesia
Neosynephrine
Sudden, severe anoxia
Carotid sinus reflex
Celiac plexus reflex
Chloroform, ethyl chloride
Cyclopropane

Rise in Blood Pressure

Position of patient
head down
lithotomy
Oxygen want, early
Carbon dioxide excess
Manipulation of toxic thyroid
Analeptics
Fluids

Fall in Blood Pressure

Position of patient
sitting
change during anesthesia
prolonged Trendelenburg
Oxygen want, late
Marked carbon dioxide excess
Deep anesthesia

Decreased Pulse Rate

Heart block
 Vomiting
 Drainage of cerebrospinal fluid
 Procaine reaction

Fall in Blood Pressure

High spinal anesthesia
 Avertin, i.v. barbiturate
 Vomiting
 Carotid sinus, celiac plexus
 Prolonged operative trauma
 Sympathectomy for hypertension
 Hemorrhage
 Accidental i.v. procaine
 Sudden release of increased
 abdominal pressure: pregnancy,
 ascites, cysts

Decreased Pulse Pressure

Carotid sinus reflex
 Celiac plexus or other traction reflexes
 Shift of mediastinum: thoracic surgery
 High spinal anesthesia
 Intracranial pressure changes
 Intracranial manipulation

CENTRAL NERVOUS SYSTEM

A reversible and controllable depression of the nervous system is the aim of any anesthesia. It would be desirable to limit the action of the drugs to the nervous system alone, but by most technics this is impossible, since the drugs are carried by the blood stream. Only by regional blocks (spinal, nerve blocks, local infiltration) are parts of the nervous system anesthetized directly.

The inhalation agents cause an ascending paralysis of the spinal cord. This means that the intercostal muscles, supplied by the thoracic cord are paralyzed before the diaphragm, with its cervical innervation. During deepening anesthesia, the lower, then the upper intercostals and finally the diaphragm lose their activity and tone, and during recovery from deep anesthesia, these muscles recover in the reverse order. Observation of the comparative activity of these two groups of muscles gives us one of our most important respiratory signs.

Concomitant with the ascending cord paralysis, depression of the brain progresses from its most specialized to its least specialized centers. The cerebrum is depressed first, with early loss in judgment and reason, followed by loss of memory and consciousness. Of the special senses, hearing remains intact the longest, and recovers first. The cerebellum then shares in the depression as evidenced by the ataxia of second stage. Objective signs of further depression are not obvious, but progress down through the thalamus, midbrain and medulla. Severe medullary depression indicates an approaching irreversibility of the anesthesia.

The most serious depressant for the nervous system is oxygen lack. In normal persons, it is a matter only of minutes (cerebrum, 8 min.; medulla, 30 min.) before permanent destruction takes place. In a person who is already in a chronic hypoxic state (cardiac, pulmonary pathology, anemia, prolonged partial obstruction of airway) this time interval is reduced to seconds. After a severe hypoxic episode, permanent sequelae, such as personality changes, visual disturbances, and changes in muscle tonus, sensation and control of fine movements may occur. Cerebral hypoxia in the newborn is known to be related to certain types of mental deficiency and spastic states appearing during the child's development. An episode of hypoxia severe enough to be incompatible with recovery is characteristically followed by failure to regain consciousness, restlessness, convulsions, hyperthermia, coma and death. Since no treatment can change this picture once it develops, prevention of oxygen want is the only means of controlling this problem.

AUTONOMIC NERVOUS SYSTEM

The autonomic nervous system is one of the most complicated and one of the most fascinating concepts in physiology. For a clear presentation of the

problem, especially of that part relating to chemical mediation of the nerve impulse, Chapter 19 in Goodman and Gilman, is recommended.

None of the anesthetic drugs fits entirely into an adrenergic or cholinergic classification but the common agents can be divided roughly as follows:

Adrenergic stimulation: ether, chloroform, oxygen lack, CO₂ excess

Cholinergic stimulation: cyclopropane, barbiturates, morphine

Spinal or nerve blocks paralyze the sympathetic fibers to the part anesthetized

Atropine and scopolamine depress cholinergic activity

Although a patient may be anesthetized to various stages of depression, stimuli are not prevented from reaching the nervous system and producing responses. Most of these reflexes are respiratory and circulatory in nature and are mediated by the autonomic nervous system. Correctly speaking, this is solely an effector system, but many afferent fibers run in autonomic nerve trunks (splanchnic, vagus). The cell bodies for these fibers are in the dorsal root ganglia, or the vagus nucleus.

Origin of afferent impulses:

Traction on upper abdominal viscera, mesentery, cecum, pelvic organs, perineum

Manipulation of recurrent and superior laryngeal nerves, phrenic nerves, vagi

Periosteal stimulation - especially rib periosteum

Lung hilum stimulation

Stimulation of celiac plexus: manipulation of common duct, pads, retractors, elevated kidney rest

Stimulation of carotid sinus: neck infections, head down position, retractor in thyroid surgery, neck dissections, anesthetist's finger pressure, extreme lateral flexion of neck

Dilatation of rectum, vagina, cervix

Overdistention of lungs

Irritation of nasal or tracheal mucous membranes

Marked apprehension preoperatively; marked excitement stage

The efferent effects may be:

Respiratory: laryngospasm, or adduction of vocal cords apnea, irregular respiration, hyperpnea, bronchospasm

Circulatory: change in pulse rate, arrhythmia, hypotension, diminution in pulse pressure, hypertension

Increase in muscle tonus, convulsions

Treatment is mainly prophylactic

1. Deep anesthesia before traction is exerted
2. Production of as little trauma as possible, and if necessary gently and steady, not sudden traction
3. Adequate premedication to control psychic state and to diminish irritability of the nervous system (morphine)
4. Endotracheal airway for upper abdominal and neck surgery
5. Blocking of area of reflex activity with procaine

After reflex has occurred, treatment may be:

1. Stopping trauma or traction when possible: reexerting traction slowly and steadily
2. Blocking the region with procaine locally
3. Changing level of anesthesia, giving adequate oxygen, and removing excess CO_2
4. Supplementing light anesthesia with additional doses of morphine (with scopolamine or atropine) for general reflex depression
5. Use of some drug to change the balance in the autonomic system: atropine, ephedrine, physostigmine, ergotamine: all have been successful in some cases

PREMEDICATION

Premedication is as important in anesthesia as the choice of agent or technic. The reasons for using premedication are several:

1. Psychic depression of patient: Abolition of undue alertness and anxiety reduces the incidence of excitement during induction, and increases the ease with which subsequent anesthetics may be given to the same person. A stormy induction usually leads to hyperactive reflexes and increased secretions, consequently to a stormier maintenance and recovery.

2. To lower patient's reflex irritability, which parallels his metabolic rate. Less anesthetic agent in actual amount is needed to produce and maintain surgical anesthesia if the starting point of the anesthesia is at a relatively low metabolic plane. Excretion of the agent and recovery from anesthesia is

consequently more prompt. If reflex irritability is depressed by premedication, there is less likelihood of such complications as vomiting during induction, breath holding and coughing with irritating agents, and reflex laryngospasm.

3. To prevent excessive secretions in the respiratory tract. The development and retention of excessive secretions during anesthesia increases the ever-present hazard of post-operative pulmonary complications. It is only because of the stimulation of secretions by ether that the term "ether pneumonia" was coined. This misnomer is commonly applied to what starts as an atelectasis and later develops into pneumonia.

4. To prevent the convulsant action of drugs used for local, block or spinal anesthesia. According to experimental evidence the barbiturates are the most effective.

5. To counteract autonomic nervous system effects of certain anesthetic agents; i.e., intravenous barbiturates and cyclopropane are apparently cholinergic drugs and are best preceded by scopolamine or atropine.

TYPES OF DRUGS USED:

Barbiturates are best for psychic depression, and for counteracting the convulsant effects of the regional drugs. They are not satisfactory for lowering reflex irritability. Alone, they cause increased secretions.

Morphine is the best drug for general metabolic depression and ~~diminution~~ of reflex irritability. Demerol is almost as satisfactory and has the added advantage of producing bronchodilation, less respiratory center depression and less nausea and vomiting than comparable doses of morphine.

Atropine and scopolamine are both good drugs for preventing secretions, counteracting the respiratory depression and the tendency to vomit after morphine, and preventing excessive parasympathetic activity. The only important difference between the two drugs is their opposite effect on the cerebral centers:

atropine is stimulating, causing increased alertness, while scopolamine is a mild depressant causing drowsiness, and sometimes amnesia.

Avertin is a premedication and not an anesthetic drug. It is desirable from the patient's point of view because of the ease with which it produces unconsciousness, but is undesirable because of the length of time protective (cough) reflexes are depressed. Used, however, only to produce amnesia, most of the objections to the drug are outweighed.

Intravenous barbiturates also produce prompt and easy loss of consciousness but in moderate doses are preferable to avertin since they are metabolized so quickly that laryngeal and cough reflexes return rapidly.

DOSE

The dose of these drugs depends upon certain factors in the patient's pathology and physiology.

The following conditions are common ones demanding more than average doses of depressant drugs:

Fever	Hyperactive personality
Pain	Muscular constitution
Marked apprehension	Chronic alcoholism
Hyperthyroidism	Pregnancy
Neurosis	Adolescence
Manic type of psychosis	Determination to resist sleep

The following demand less than average doses:

Anemia	Nephritis
Hemorrhage	Senility, infancy
Shock	Previous depressant drug
Hypothyroidism	Jaundice
Chronic illness	Mental deficiency
Weight loss	Schizophrenia
Acute alcoholism	Senile psychosis
Cardiac disease	Phlegmatic personality
Obesity	Addison's disease

ROUTE

The route of administration of these drugs depends on the time available before start of anesthesia, since it is desirable that the peak of action

of the premedication be reached or just passed at the time anesthesia is begun. When the medication is given hypodermically 90 minutes is necessary for the development of maximum effect, when intramuscularly, 45-60 minutes, while only 10 to 15 minutes are needed for the peak of action after intravenous premedication. The last is, therefore, the only efficacious route when the time between scheduling an operation and the start of anesthesia is short. The oral and rectal routes are both undependable because of the many variable factors in absorption.

The following generalities are suggested for medication before various anesthetic agents:

Nitrous oxide needs heavy premedication. Nitrous oxide is not a potent agent, therefore the metabolic rate and reflex irritability must be brought to "basal conditions" by appropriate doses of morphine or by a "basal anesthetic" (Avertin) in order to produce surgical anesthesia without sacrificing adequate oxygenation. Reduction of reflex irritability and metabolic rate not only lessen the amount of nitrous oxide necessary, but also tend to reduce the oxygen requirements of the tissues.

Cyclopropane is potent and rapid in effect, therefore need not be preceded by the depressant type of medication, but because it is cholinergic atropine or scopolamine should be administered to counteract this effect.

Ether, because of its slow onset and unpleasant irritating qualities should be preceded by moderate doses of the depressant drugs and sufficient atropine or scopolamine to inhibit excess secretions.

Local, spinal or regional technics are best preceded by a short acting barbiturate, and a small dose of morphine, but inhibition of secretions is not necessary unless the use of supplementary anesthesia (ether, cyclopropane, i.v. barbiturates) is anticipated. Scopolamine is frequently used before local, block or spinal anesthesia, but it is used for its counteracting of respiratory

depression and nausea from morphine, and for its production of amnesia, rather than for its effect on secretions.

Intravenous barbiturates, like cyclopropane are cholinergic, and are frequently accompanied by increased secretions. Atropine or scopolamine should therefore be given before their use. Small doses of morphine to reduce pain perception are helpful since responses to pain stimuli are abolished only with relatively deep barbiturate anesthesia. Oral premedication with small amounts of other barbiturates is not contraindicated, but is usually unnecessary and best omitted.

Avertin is essentially a depressant premedication, and so should be preceded by minimal doses of morphine (for reflex depression) but the necessary amount of atropine or scopolamine should not be reduced.

Suggested doses in healthy adults are

Magendie 0.3 c.c. (morphine .009 gm) + Scopolamine 0.0004 gm.

Demerol 0.075 gm + Scopolamine 0.0004 gm.

General principles of medication:

1. A combination of two depressant drugs, i.e., nembutal and morphine is more depressant than either one alone. Correct this by decreasing the dose of each.
2. Because the combination of barbiturate, morphine and a general anesthetic so often produces marked respiratory depression, it is better to give the barbiturate the night before operation, using a medium or long acting one so that its action will be present but waning on the morning of operation. For cases scheduled late in the morning a short-acting barbiturate (nembutal, seconal) given "on awakening" will allay anxiety, yet be past its peak of action when the pre-operative morphine is given.
3. Beware of using depressant drugs in patients with preexisting

respiratory difficulty. Such patient's respiratory exchange may be just adequate to keep them oxygenated without such drugs, but even slight respiratory depression caused by the drugs may lead to severe hypoxia. Patients with severe hemorrhage, anemia, asthma, cardiac decompensation, substernal thyroids, marked kyphosis, and marked abdominal distention, are in this group, as well as those with pulmonary disease.

4. Every time a depressant drug is used, the cough reflex is diminished. The dosage of opiates necessary to control cough is materially smaller than that necessary to relieve pain. An active cough reflex is the best prophylaxis against respiratory complications. THINK TWICE before ordering depressant drugs.

INHALATION AGENTS

There are at least ten anesthetic gases and vapors. A comparison of important properties of the six most common is tabulated on page 19.

The anesthetic gases obey all the well-established physical gas laws, but the one of particular importance to the physiology of anesthesia is that the direction of diffusion of a gas is from a region of high tension (partial pressure) to regions of lower tensions. This is the mechanism by which gases pass from the alveoli to the blood stream and thence to the tissues when they are administered, and pass in the reverse direction to be excreted when administration is stopped.

An important difference between the volatile agents and the non-volatile ones is that they are not changed in the body, and are quantitatively excreted by the lungs. A negligible amount diffuses through the skin and into the sweat, urine, and other secretions, but the liver and kidneys are spared the metabolic work of inactivating the drugs. The significance of this mode of excretion is that should overdose occur, the drug can be removed from the body by

SOME PROPERTIES OF COMMON INHALATION ANESTHETIC AGENTS

NAME	ETHER	CHLORO- FORM	VINETHENE	CYCLO- PROPANE	ETHYLENE	NITROUS OXIDE
FORMULA	$\begin{array}{c} \text{C}_2\text{H}_5 \\ \diagup \quad \diagdown \\ \text{O} \\ \diagdown \quad \diagup \\ \text{C}_2\text{H}_5 \end{array}$	CHCl_3	$\begin{array}{c} \text{CH}=\text{CH}_2 \\ \diagup \quad \diagdown \\ \text{O} \\ \diagdown \quad \diagup \\ \text{CH}=\text{CH}_2 \end{array}$	$\begin{array}{c} \text{CH}_2 \\ \diagup \quad \diagdown \\ \text{CH}_2 \quad \text{CH}_2 \end{array}$	$\text{CH}_2=\text{CH}_2$	N_2O
STABILITY	FAIR	FAIR	POOR	GOOD	GOOD	GOOD
SP.GR. VAPOR/GAS (Air=1)	2.55	4.1	2.42	1.45	.97	1.52
FIRE & EXPLOSION HAZARD	++++	0	++++	++++	++++	0
POTENCY WITHOUT OXYGEN WANT	100%	100%	90%	90%	40%	25%
% IN INSPIRED AIR (2nd plane)	4.0%	1.5%	4.0%	20%	80-85%	80-90%
RAPIDITY OF INDUC- TION - CONTROL & RECOVERY	+	++	++	++++	++++	++++
MARGIN OF SAFETY	Depends on ability of anesthetist					
INCR. SECRETIONS	++++	+++	++++	+	+	0
ADVERSE METABOLIC CHANGES	+++	++++	+++	++	+	0
CAUSE OF DEATH	RESP. DEPRESS. ANOXIA	CARDIAC DEPRESS. ANOXIA	RESP. DEPRESS. ANOXIA	RESP. DEPRESS. ANOXIA	ANOXIA	ANOXIA

Curare preparations administered intravenously can be used to produce relaxation of striated muscle and is especially indicated with the less potent anesthetic agents. Some respiratory depression always accompanies its use and some method of assisted ventilation should always be at hand to assure proper exchange of gases.

artificial respiration provided that the circulation has not entirely ceased to carry the drug from the tissues to the lungs. There is more safety in using a drug which can be removed in the same way it is administered, than in using a drug which must be metabolized by the patient before lightening of the anesthesia can occur.

Many theories (see Goodman and Gilman, p. 32) have been advanced to explain the phenomenon of narcosis, but few are equally applicable to all types of anesthetic drugs. It is most likely that the ultimate mechanism of the production of narcosis is an interference with the oxidative enzyme systems of nervous tissue. The oil-water solubility ratio of the anesthetic drug, its adsorptive properties, or its alteration of surface tension may be of importance only in intermediate steps leading to the alteration of tissue oxidation. This assumption explains why several of the theories may have considerable corroborative evidence without being sufficiently applicable to all drugs to permit their acceptance. It is far from established at the present time that enzyme disturbances are the explanation of reversible cell depression (narcosis), but a shift in the relative importance of different enzyme systems is a tempting way of explaining how nerve cell activity (energy production) can be inhibited by anesthetic drugs without causing death of the cell.

INHALATION TECHNIQS

Inhalation methods as previously mentioned are the only ones in which the anesthetic drug can be removed from the patient at will, and thereby are the safest of all methods. They also allow the most accurate individualization of dosage.

The following factors directly influence inhalation anesthesia:

Change in the inspired atmosphere
 Minute volume exchange
 Available alveolar surface
 Patient's respiratory tract

Efficiency of the circulation
 Blood supply to various tissues
 Amount of adipose tissue in body
 Composition of the blood

There are three fundamental types of inhalation technics, with numerous variation; open drop, carbon dioxide absorption, and insufflation technics.

The open drop method is characterized by the use of a wire mask covered thinly with layers of gauze, on which the liquid anesthetic agent is dropped and allowed to vaporize. The vapor being heavier than air, concentrates under and close to the mask, and is inhaled by the patient. Exhalation through the mask increases the rate of vaporization of the liquid.

The carbon dioxide absorption system involves the use of a machine with yokes for attaching tanks of compressed gases, valves to adjust the rate at which the gases escape from the tanks, a flowmeter to indicate the flow of gas, a rubber bag to act as a reservoir for the gas mixture, and a canister of absorbing material (sodalime, baralyme) to absorb the carbon dioxide made by the patient.

Insufflation methods are characterized by blowing an anesthetic mixture into the patient's respiratory tract (naso-pharynx, mouth or trachea) in sufficient concentrations to maintain surgical anesthesia, after it has been induced by one of the above methods. The anesthetic mixture may be composed of gases delivered from high pressure tanks at a more rapid rate of flow than used with the closed system, and may be used as vehicles to carry the vapor of a liquid drug from a reservoir to the patient. (Examples: Insufflation of nitrous oxide and oxygen, ether vapor carried by a stream of oxygen.) Compressed air piped to the operating room from a central source, or delivered by a motor-driven pump or foot bellows, may be used instead of oxygen as the vehicle for the vapor of volatile agents.

All three systems can be considered as extensions of the respiratory

system of the patient. The gases under the mask, in the breathing bag, or in the insufflation stream come into equilibrium with the alveolar gas concentrations after a short time. From this point, equilibrium is reached with the blood stream, and finally with the tissues.

There are advantages and disadvantage of each method:

<u>Open drop</u>	<u>PRO</u>	<u>CON</u>
1.	Simple, portable	1. No control of respiration
2.	Inexpensive equipment	2. Poor set-up for resuscitation
3.	Minimal rebreathing CO ₂	3. Very wasteful, anes. agent
4.	Minimal resistance to breathing	4. Loss of heat, and water vapor
		5. Irritating, cold vapor
		6. Always lower oxygen than air
		7. Fire hazard
		8. Skin burns, liquid ether
<u>Absorption</u>		
1.	Good control respiration	1. Initial expense high
2.	Resuscitation easy	2. Less portable; tanks of gases
3.	Economical, anes. agent	3. Can get out of order
4.	Heat, water vapor preserved	4. Increased temperature inspired air
5.	Oxygen easily added, carbon dioxide removed	5. Slight resistance to breathing
		6. Explosion hazard
<u>Insufflation</u>		
1.	Technically easy, head and neck surgery	1,2,3,4,5, and 7 same as open drop method
2.	Lowest rebreathing CO ₂	1 and 3 same as absorption
3.	Easy route to add O ₂	Relatively portable when foot bellows type used
		Difficult to maintain deep anesthesia

To each system, an endotracheal airway, introduced through the nose or mouth, may be added. Its advantages and disadvantages are listed below:

<u>PRO</u>	<u>CON</u>
1. Free airway, common sites of obstruction under control (tongue, larynx)	1. Moderate obstruction to respiration if tube too long or narrow
2. Dead space diminished - quieter respiration, lower CO ₂	2. Possible trauma to larynx and pharynx by inexperienced anesthetist
3. Prevention of material from entering trachea (tube with cuff or packing)	3. Possible injury to epithelium of trachea or larynx by pressure of tube or cuff
4. Pathway for suction established	4. Anesthesia must be kept deep enough to abolish cough reflex
5. Control of pressure in chest	
6. Ideal set-up for resuscitation	

Indications for endotracheal airway

1. Operations in areas of high reflex irritability: upper abdomen
2. Operations on patients with high reflex irritability: alcoholics
3. Head and neck surgery: anesthetist out of surgeon's way, but good control of airway
4. Poor risk patients: if resuscitation may be necessary; especially neurological patients
5. Thoracic surgery: control of pressure relations in chest; suction of secretions from trachea and bronchi
6. Insufflation technics, delivery of agent more efficient and effective
7. Long operations in unphysiological positions: Ex.: spinal fusion, in prone position. Respirations often aided by anesthetist
8. Patients in whom an adequate airway cannot be established and maintained by simpler means: receding jaw, short neck, intractable laryngospasm
9. In war surgery when one anesthetist must manage several general anesthetics at once

OTHER TECHNIQUES

Rectal, Intravenous, Regional Blocks

In these technics, drugs can be administered with comparative ease, but cannot be recovered at will. The patient destroys and excretes the metabolized drug at varying and uncontrollable rates of speed.

PRO

CON

Rectal route

Simple, portable
Painless
No fire hazard
(except ether)

Unpredictable absorption
Variation in rectal preparation
Sphincter relaxation
Needs cooperative patient
Rectal pathology
Long recovery period, requiring constant nursing care

Intravenous

Simple, portable
Minimal pain
No fire hazard
Quick induction
Quick recovery
Intermittent dosage quite accurate
Good supplement for blocks

Narrow margin of safety
Overdose of drug easy
No control of respiration
Only light anesthesia obtainable
May need help to keep free airway (two people desirable)

Nerve Blocks

Excellent relaxation
 No fire hazard
 Portable
 Minimal metabolic
 disturbance because
 of limited area
 of anesthesia

Special knowledge and training
 Limited duration of block
 Need cooperation (children
 difficult)
 Possible damage to nerve

Avertin overdose results in marked circulatory and respiratory depression (more of depth than rate). Treat with pressor drugs (ephedrine, neosynephrine) and oxygen.

Pentothal overdose results in apnea with variable degrees of circulatory depression. Treat with artificial respiration with oxygen. Analeptic drugs (coramine, benzedrine, metrazol, picrotoxin) are advisable only when overdose is known to be large since smaller doses of pentothal may be metabolized quickly and the patient then may have an overdose of the analeptic.

Procaine "reaction" from overdose or intravenous injection results in one of two syndromes:

1. Marked blood pressure drop, bradycardia, pallor, nausea and vomiting. Treat with pressor drugs (ephedrine, neosynephrine) and oxygen.

2. Increased C.N.S. stimulation: apprehension, restlessness, convulsions and hypoventilation from spasm of respiratory muscles. Treat with intravenous barbiturate and oxygen (artificial respiration if necessary). Prevent by proper premedication.

SPINAL ANESTHESIAPRO

Simple technic, portable
 Non-explosive
 Excellent relaxation
 Minimal metabolic changes

CON

Numerous uncontrollable factors
 Drug cannot be recovered
 Circulatory depression frequent
 Possible nerve root damage
 Late recovery of active intercostals
 Shock more severe after hemorrhage

AVERTIN			PENTOTHAL	PROCAINE
Synonym	Tribromethanol in Amylene hydrate		Thio nembutal	Novocaine, neocaine
Route	Rectal		Intravenous	Nerve Blocks, spinal
Stability	Good in dark bottles		Good dry in ampoules 12 hours in solution	Good
Respiratory effects	Depressed tidal volume		Markedly depressed tidal volume	None
Circulatory effects	Blood pressure drop decreasing cardiac output		Blood pressure drop with deep or rapid anesthesia	Occasional blood pressure drop
Potency	Satisfactory for premedication; anesthetic only in overdose		Fair--(1st plane)	Good
Liver & kidney function	Moderately decreased		Not affected	Not affected
Excretion	Liver and kidneys		Unknown site of destruction. End product in urine	Liver and kidneys
Contraindications	Hepatic and renal disease, shock, anemia, extremes of blood pressure, extremes of age, low vital capacity		Renal disease, shock, anemia, extremes of blood pressure, extremes of age, low vital capacity, asthma	None, except rare sensitivity to drug

Factors concerned in the DURATION and HEIGHT of spinal anesthesia

<u>Controllable</u>	<u>Uncontrollable</u>
Space of injection	Diffusion
Speed of injection	Circulation of spinal fluid
Volume of solution used	Movement of fluid by respirations and pulsations
Dosage of drug	Length of spine
Specific gravity sol.	Previous pathology or anomalies
Position of patient	Movement of patient
Choice of drug	Change CSF pressure by straining
	Breaking needles during administration

DRUGS used:

Procaine.....short acting, but least toxic
 Monocaine.....isomer of procaine
 Metycaine.....longer acting than procaine
 Pontocaine....long acting, 10X more toxic than procaine
 Nupercaine.... " " , 20X " " " "

Glucose or alcohol may be added to the first four drugs to make the solution hyper- or hypobaric. Nupercaine is slightly hyperbaric in the 1:200 solution, and may have glucose added; it is hypobaric in the 1:1500 solution.

Continuous or intermittent method of administration allows for much more accurate dosage, and assures adequate anesthesia until end of operation. Severe respiratory depression from too high a level can be avoided entirely. Circulatory depression, however, has not been avoided by the continuous method.

Some of the factors in the production of circulatory depression by spinal anesthesia during surgery, are listed below:

1. Relaxation of skeletal muscles, followed by poorer venous return.
2. Vasodilatation of blood vessels in the anesthetized regions.
3. Stagnation of blood in the peripheral vascular bed.
4. Diminished thoracic excursion from partly paralyzed intercostal muscles causing disturbance of pressure relations and poor venous return to heart.
5. Hypoxia of myocardium.
6. "Toxic" effect of anesthetic drug itself.
7. Accentuated vagotonic response to traction, due to autonomic imbalance resulting from anesthetized splanchnic nerves.
8. Poor adjustment to change in position.
9. Poor compensation to changes in blood volume (hemorrhage).
10. Decreased adrenalin output. (Adrenals functionally denervated.)

Treatment is best prophylactic, by use of a vasopressor drug, which may act at one of three sites, the vasomotor center, the heart muscle, or on the blood vessels peripherally. The best vasopressor drug is ephedrine, because of its sustained action, and minimal toxic effects. Other drugs in use for this purpose are neosynephrine, benzedrine, cobefrin and paredrine.

After the drop in pressure has occurred, treatment should be directed toward providing ample oxygenation for the brain and heart muscle.

1. Head down position (if hyperbaric drug has not been added to subarachnoid space recently)
2. Oxygen, by insufflation or closed inhalation system
3. Restoration of normal ventilation by manual methods
4. Additional doses of pressor drug
5. Fluid therapy: blood, plasma or saline, as indicated

Retching is a common and troublesome complication of spinal anesthesia.

It may be caused by:

1. Intraabdominal traction
2. Hypoxia due to:
 - a. Marked blood pressure fall
 - b. Respiratory paralysis
 - c. Overdose premedication
3. Reaction to premedication (morphine)
4. Psychic
 - a. Inadvertent seeing of blood on drapes
 - b. Odor of "prep" ether - odor of cautery
 - c. Anxiety
5. Reaction to spinal drug (only with very large doses)

Treatment of retching under spinal anesthesia depends on the cause.

- For 1: Exert traction as slowly and gently as possible. Depress reflex irritability with morphine or light general anesthesia.
- For 2: Supply oxygen: artificial respiration with mask and bag if tidal volume is too small or paralysis complete.
- For 3: Scopolamine or atropine may help. (1 part:25 parts morphine) and treat as for #4.
- For 4: Any treatment that distracts patient will help: deep breathing, ice water compress to forehead, aromatic spirits of ammonia, etc.
- For 5: As for procaine overdose.

POSTOPERATIVE PULMONARY COMPLICATIONS

Pulmonary complications following anesthesia and surgery form the most frequent and most serious type of morbidity and mortality. Many factors contribute to their incidence, but most revolve around the ineffectual removal of secretions from the tracheobronchial tree. Under normal conditions, secretions are constantly produced in this tract, are in part dried by the air currents, and in part removed by the action of the ciliated epithelium, as well as by the peristaltic action of the bronchial musculature. Both these actions are inhibited by anesthetic agents, and by therapeutic doses of morphine. The defense of the patient against accumulated secretions is further weakened by the presence of an ineffective cough. The following circumstances contribute to its ineffectiveness.

1. Inability to inspire deeply
 - Splinting of abdominal muscles inhibiting motion of diaphragm
 - Paralysis or paresis of intercostals following spinal anesthesia
 - Fluid or air below diaphragm, or in chest
 - Tight binders
 - Prone, or lateral position
 - Phrenicectomy
 - Bronchospasm
2. Inadequate cough reflex
 - Too much postoperative medication (morphine)
 - Irresponsive central nervous system
 - Weak, exhausted patient, senility
3. Inability to tighten abdominal muscles
 - Post-spinal paresis
 - Ventral herniae, diastasis
 - Avitaminosis
4. Inability to produce positive pressure in chest
 - Defect in chest wall (thoracoplasty)
 - Open glottis (endotracheal tube); tracheotomy

The patient most likely to develop postoperative pulmonary complications are those with:

1. Previous history of pulmonary complication, especially postoperative pneumonia
2. Chronic respiratory infection, with sputum: chronic bronchitis and emphysema
3. Upper abdominal operations
4. Neurological operations
5. Long period of immobility in immediate postoperative period: plaster cast, or poor nursing care

6. Alcoholic history
7. Operations of over three hours' duration
8. Presence of shock during operation or in postoperative period

The two types of pathology which develop are:

1. Atelectasis: patchy or confined to one lobe, or one lung. The origin of the atelectasis is most frequently obstruction by mucous plugs lodged in the major bronchi, or in smaller divisions of the bronchi. The obstruction may also be caused by reflex bronchospasm. The gases in the lung distal to the obstruction are absorbed into the blood stream, the alveoli become airless, and the solid area of lung thus produced is a perfect field for the development of infections, or

2. Bronchopneumonia: the causative organisms of this complication may be those present in the lower tract before operation, or ones aspirated during anesthesia. Aspiration of vomitus almost assures development of pneumonia, not so much because of the bacteria present in this instance, but because the chemical irritation from the hydrochloric acid predisposes to infection.

Prophylactic treatment before operation consists of

1. Preventing secretions by use of belladonna group.
2. Minimal use of belladonna group if secretions already present.
3. Postural drainage to remove sputum; occasionally bronchoscopy.
4. Afternoon operations for thoracic surgery.
5. Choice of non-irritating anesthetic agent.
6. Good oral hygiene.
7. Empty stomach before operation.

Prophylactic treatment at end of operation consists of

1. Presence of cough reflex before patient leaves operating room.
2. Removal of secretions by aspiration, and stimulation of cough reflex.
3. Minimal postoperative medication for pain relief.
4. Hyperventilation, by voluntary deep breathing, or by carbon dioxide inhalations (100% air) if patient uncooperative.
5. Frequent change in position.
6. Head down position until patient conscious.
7. Intercostal block in upper abdominal cases.
8. Use of non-diffusible gas at end of anesthesia (air or helium).

Treatment if atelectasis occurs:

1. Change in position, with affected lung uppermost.
2. Hyperventilation in several positions.
3. Assistance with expiration by judicious pounding on chest.
4. Aspiration of trachea with catheter, and stimulation of cough reflex.
5. Bronchoscopy, if above are inadequate.
6. Penicillin therapy.

EXPLOSION HAZARD IN ANESTHESIA

The incidence of explosions with ethylene, cyclopropane and ether is roughly between 1:100,000 and 1:500,000 cases. Statistically this is one of the most unimportant anesthesia complications, but it should be zero.

The controversy between safety of a completely grounded operating room vs. room completely isolated from ground still continues. Other questions under investigation involve the use of conductive rubber, the use of inert gases with explosive gases to make them non-explosive, and the true nature of static electricity. Air conditioning, by raising the humidity in the operating room, permits partial distribution of static charges so that sparks are less apt to pass between points of different potentials. If the atmosphere has been washed free of CO₂ by the air conditioning, however, even very humid air will not prevent sparks.

All the inhalation agents except nitrous oxide and chloroform are explosive in the ranges in which we use them. A few common sense principles apply to their use:

1. If the use of electrical equipment is imperative for the surgery, choose a non-explosive agent: nitrous oxide, with ample premedication; intravenous barbiturate, spinal, nerve block or local infiltration, or combinations of these technics.
2. If explosive agents are being used, avoid using electrical equipment; i.e., carbohc knife for severing appendix instead of cautery.

3. The danger zone with these drugs, is at and near the face mask. It is the mixture leaking at the face mask which is hazardous, not blood or tissues containing the agent. If electrical equipment must be used at a distance from the face, partial protection may be afforded by keeping fit of the mask as tight as possible, or enclosing the area in a wet towel.

4. It is believed widely that danger is minimized if the patient, machine and anesthetist are kept at the same electrical potential by using an inter-coupler between these three. This protects only against static electricity, and is entirely useless for sparks from a cautery, coagulating machine, or X-ray apparatus, and similar electrical equipment.

5. Members of the operating room staff, visitors and bystanders should avoid brushing past the anesthesia machine or bumping it with metal objects, such as spotlights, and portable tables.

6. Known sources of static electricity, such as wool blankets and wool, silk or rayon outer garments should be prohibited from the operating room.

RESUSCITATION

A few general principles apply to any type of resuscitation done for any reason by any of a number of technics.

1. There must be a FREE AIRWAY. Artificial respiration is futile unless the airway is patent.

Explore mouth with finger, remove foreign bodies.

Remove fluids by gravity: head down position.

Elevate chin, turn in lateral or prone position to keep tongue from obstructing airway.

Introduce pharyngeal or endotracheal airway if available.

Do tracheotomy, if above not available.

2. The patient must be ventilated. There is no way to store oxygen. It must be supplied immediately. Prone pressure method, or mouth to mouth respiration are always available.
3. The patient is in shock. Warmth and head down position should be supplied.
4. Stimulating drugs are of NO USE unless the patient is well oxygenated.